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NEWS 17 JUL 28 CA/CAPLUS patent coverage enhanced
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NEWS 22 AUG 13 CA/CAPLUS enhanced with printed Chemical Abstracts
page images from 1967-1998
NEWS 23 AUG 15 CAOLD to be discontinued on December 31, 2008
NEWS 24 AUG 15 CAPLUS currency for Korean patents enhanced
NEWS 25 AUG 25 CA/CAPLUS, CASREACT, and IFI and USPAT databases
enhanced for more flexible patent number searching
NEWS 26 AUG 27 CAS definition of basic patents expanded to ensure
comprehensive access to substance and sequence
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=> S (common gamma chain) or (gammac) (4A) (cytokine receptor)
L1 1753 (COMMON GAMMA CHAIN) OR (GAMMAC) (4A) (CYTOKINE RECEPTOR)

=> S (cytokine receptor) (4A) (structure)
L2 120 (CYTOKINE RECEPTOR) (4A) (STRUCTURE)

=> S (cytokine receptor) (4A) (function or activity or disease)
L3 278 (CYTOKINE RECEPTOR) (4A) (FUNCTION OR ACTIVITY OR DISEASE)

=> s l1 and l2 and l3
L4 1 L1 AND L2 AND L3

=> d l4 bib ab

L4 ANSWER 1 OF 1 CAPLUS COPYRIGHT 2008 ACS on STN
AN 1996:618362 CAPLUS
DN 125:325571
OREF 125:60975a,60978a
TI Common .gamma. chain in X-linked severe
combined immunodeficiency
AU Mayumi, Mitsufumi
CS Fac. Med., Kyoto Univ., Kyoto, 606-01, Japan
SO Saishin Igaku (1996), 51(10), 1772-1776
CODEN: SAIGAK; ISSN: 0370-8241
PB Saishin Igakusha
DT Journal; General Review

LA Japanese
AB A review with 11 refs., on the structure and function
of common cytokine receptor γ chain, γ
chain gene mutation in X-linked severe combined immunodeficiency (X-SCID),
and diagnosis of X-SCID by γ chain gene.

=> S 11 and 12
L5 1 L1 AND L2

=> d 15 bib

L5 ANSWER 1 OF 1 CAPLUS COPYRIGHT 2008 ACS on STN
AN 1996:618362 CAPLUS
DN 125:325571
OREF 125:60975a,60978a
TI Common .gamma. chain in X-linked severe
combined immunodeficiency
AU Mayumi, Mitsufumi
CS Fac. Med., Kyoto Univ., Kyoto, 606-01, Japan
SO Saishin Igaku (1996), 51(10), 1772-1776
CODEN: SAIGAK; ISSN: 0370-8241
PB Saishin Igakusha
DT Journal; General Review
LA Japanese

=> S (cytokine receptor) (4A) (disease)
L6 112 (CYTOKINE RECEPTOR) (4A) (DISEASE)

=> s 11 and 16
L7 6 L1 AND L6

=> duplicate
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=> d 18 1-6 bib ab

L8 ANSWER 1 OF 6 CAPLUS COPYRIGHT 2008 ACS on STN
AN 2002:965107 CAPLUS
DN 138:38085
TI Cytokine receptor common gamma chain like
(CRGCL), polynucleotides, antibodies for diagnosis and treatment of
immune disease
IN Ruben, Steven M.; Rosen, Craig A.; Moore, Paul A.
PA Human Genome Sciences, Inc., USA
SO U.S. Pat. Appl. Publ., 140 pp., Cont.-in-part of Appl. No. PCT/US00/22493.
CODEN: USXXCO
DT Patent
LA English
FAN.CNT 5

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
	-----	---	-----	-----	-----
PI	US 20020193305	A1	20021219	US 2002-78059	20020220
	US 6861227	B2	20050301		
	WO 9947538	A1	19990923	WO 1999-US5068	19990305

W: AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE, DK, EE, ES, FI, GB, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, UA, UG, US, UZ, VN, YU, ZW

RW: GH, GM, KE, LS, MW, SD, SL, SZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG

US 6844170 B1 20050118 US 1999-263626 19990305
US 20030028006 A1 20030206 US 1999-376430 19990818
US 6982320 B2 20060103
WO 2001012672 A2 20010222 WO 2000-US22493 20000817

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CR, CU, CZ, DE, DK, DM, DZ, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW

RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG

US 20040266693 A1 20041230 US 2004-899107 20040727
PRAI US 1998-78563P P 19980319
US 1998-86505P P 19980522
US 1999-263626 A2 19990305
WO 1999-US5068 A2 19990305
US 1999-376430 A2 19990818
WO 2000-US22493 A2 20000817
US 2001-269876P P 20010221
US 2002-78059 A3 20020220

AB The present invention relates to a novel human gene encoding a polypeptide which is a member of the cytokine receptor family. More specifically, the present invention relates to a polynucleotide encoding a novel human polypeptide named Cytokine Receptor Common Gamma Chain Like, or "CRGCL". This invention also relates to CRGCL polypeptides, as well as vectors, host cells, antibodies directed to CRGCL polypeptides, and the recombinant methods for producing the same. Also provided are diagnostic methods for detecting disorders related to the immune system, and therapeutic methods for treating diagnosing, detecting, and/or preventing such disorders. The invention further relates to screening methods for identifying agonists and antagonists of CRGCL activity.

RE.CNT 37 THERE ARE 37 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L8 ANSWER 2 OF 6 BIOSIS COPYRIGHT (c) 2008 The Thomson Corporation on STN
AN 2000:280032 BIOSIS
DN PREV200000280032
TI Stable and functional lymphoid reconstitution of common cytokine receptor gamma chain deficient mice by retroviral-mediated gene transfer.
AU Soudais, Claire; Shih, Tsujino; Sharara, Lama I.; Guy-Grand, Delphine; Taniguchi, Tadatsugu; Fischer, Alain; Di Santo, James P. [Reprint author]
CS Unite des Cytokines et Developpement Lymphoide, Institut Pasteur, 25 Rue du Dr Roux, F-75 724, Paris, France
SO Blood, (May 15, 2000) Vol. 95, No. 10, pp. 3071-3077. print.
CODEN: BLOOAW. ISSN: 0006-4971.
DT Article
LA English
ED Entered STN: 6 Jul 2000
Last Updated on STN: 7 Jan 2002
AB Mutations in the gene encoding the common cytokine

receptor gamma chain (gammac) are responsible for human X-linked severe combined immunodeficiency disease (SCIDX1). We have used a gammac-deficient mouse model to test the feasibility and potential toxicity of gammac gene transfer as a therapy for SCIDX1. A retrovirus harboring the murine gammac chain was introduced into gammac-deficient bone marrow cells, which were then transplanted into alymphoid RAG2/gammac double-deficient recipient mice. Circulating lymphocytes appeared 4 weeks postgraft and achieved steady-state levels by 8 weeks. The mature lymphocytes present in the grafted mice had integrated the gammac transgene, expressed gammac transcripts, and were able to proliferate in response to gammac-dependent cytokines. The gammac-transduced animals demonstrated (1) normal levels of immunoglobulin subclasses, including immunoglobulin G1 (IgG1) and IgG2a (which are severely decreased in gammac- mice); (2) the ability to mount an antigen-specific, T-dependent antibody response showing effective in vivo T-B cell cooperation, and (3) the presence of gut-associated cryptopatches and intraepithelial lymphocytes. Importantly, peripheral B and T cells were still present 47 weeks after a primary graft, and animals receiving a secondary graft of gammac-transduced bone marrow cells demonstrated peripheral lymphoid reconstitution. That gammac gene transfer to hematopoietic precursor cells can correct the immune system abnormalities in gammac- mice supports the feasibility of in vivo retroviral gene transfer as a treatment for human SCIDX1.

L8 ANSWER 3 OF 6 BIOSIS COPYRIGHT (c) 2008 The Thomson Corporation on STN
AN 2001:316794 BIOSIS
DN PREV200100316794
TI Gene therapy of human severe combined immunodeficiency (SCID)-X1 disease.
AU Cavazzana-Calvo, M. [Reprint author]; Hacein-Bey, S. [Reprint author]; de
Basile, G.; Dupuis-Girod, S.; Thrasher, A.; Wulffraat, N.; Sorensen,
R.; Casanova, J. L.; Le Deist, F.; Fischer, A.
CS Laboratoire de Therapie Cellulaire et Genique, Hopital Necker, Paris,
France
SO Blood, (November 16, 2000) Vol. 96, No. 11 Part 1, pp. 590a. print.
Meeting Info.: 42nd Annual Meeting of the American Society of Hematology.
San Francisco, California, USA. December 01-05, 2000. American Society of
Hematology.
CODEN: BLOOAW. ISSN: 0006-4971.
DT Conference; (Meeting)
Conference; Abstract; (Meeting Abstract)
LA English
ED Entered STN: 4 Jul 2001
Last Updated on STN: 19 Feb 2002
AB SCID-X1 is characterized by an absence of mature T and NK lymphocytes due
to gammac cytokine receptor deficiency.
From 03/99 to 02/00, five patients (pts) have been enrolled into a gene
therapy clinical trial. Pts age ranged from 1 to 9 months, 3 of them had
a family history of X-linked immunodeficiency, and 3 had severe
infections. Maternal T-cell engraftment was detected in two children.
SCID-X1 diagnosis was based on blood lymphocyte phenotype determination
and findings of a gammac gene mutation. CD34+ cells were preactivated,
then infected daily for 3 days with the MFG gammac-vector-containing
supernatant in bags coated with the CH296 fibronectin fragment in the
presence of SCF, M-GDF, Flt 3L and IL-3. Fourteen to 26.5 X 10⁶/kg CD34+
cells were infused back to the pts without prior chemoablation. Thirty
four to 41 % of CD34 +cells expressed the gammac transgene. In all pts
but one, T lymphocyte counts were detected from day 30 and rose
progressively to reach values gtoreq 3500/mul after 1 year for the first
two pts and appr 4800/mul for the fourth patient. T lymphocytes were
shown to be polyclonal, to express the gammac transgene to contain 1 to 3
copies of the provirus and proliferate in vitro in the presence of

mitogens, IL-2 and antigens after pts immunization. The fifth patient has 4 months follow up but transduced T are detectable (gtoreq 1000/mul). The third patient had a disseminated BCG infection with massive splenomegaly. No T cell differentiation has been detected until 6 months after injection despite improved clinical state and persistent detection of gammac+ cells in his blood as well as transduced B and NK cells in the spleen. Likely genetically modified CD34+ cells have been trapped in the spleen of this patient. In three pts, Ig perfusions have been stopped. One year, 9.5 months and 5 months after cessation of the Ig substitution specific antibodies to tetanus and diptheria toxoids as well as to polioviruses were found in their serum, together with detectable concentrations of IgG and IgM. In 4 out of five pts, evidence for full known correction of the immune deficiency has been provided. They are free of infections without therapy, doing well with follow up of 16, 14.5, 9 and 4.5 respectively.

L8 ANSWER 4 OF 6 CAPLUS COPYRIGHT 2008 ACS on STN
 AN 1999:613930 CAPLUS
 DN 131:241990
 TI Cytokine receptor common gamma chain like
 IN Ruben, Steven M.; Rosen, Craig A.; Moore, Paul A.
 PA Human Genome Sciences, Inc., USA
 SO PCT Int. Appl., 149 pp.
 CODEN: PIXXD2

DT Patent
 LA English
 FAN.CNT 5

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	WO 9947538	A1	19990923	WO 1999-US5068	19990305
	W: AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE, DK, EE, ES, FI, GB, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, UA, UG, US, UZ, VN, YU, ZW				
	RW: GH, GM, KE, LS, MW, SD, SL, SZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG				
	CA 2323776	A1	19990923	CA 1999-2323776	19990305
	AU 9930727	A	19991011	AU 1999-30727	19990305
	EP 1093457	A1	20010425	EP 1999-912330	19990305
	R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, FI				
	JP 2002506625	T	20020305	JP 2000-536731	19990305
	US 20030028006	A1	20030206	US 1999-376430	19990818
	US 6982320	B2	20060103		
	US 20020193305	A1	20021219	US 2002-78059	20020220
	US 6861227	B2	20050301		
	US 20040266693	A1	20041230	US 2004-899107	20040727
PRAI	US 1998-78563P	P	19980319		
	US 1998-86505P	P	19980522		
	US 1999-263626	A2	19990305		
	WO 1999-US5068	W	19990305		
	US 1999-376430	A2	19990818		
	WO 2000-US22493	A2	20000817		
	US 2001-269876P	P	20010221		
	US 2002-78059	A3	20020220		
AB	The present invention relates to a novel human protein called cytokine receptor common gamma chain like or CRGCL, and isolated polynucleotides encoding this protein. CRGCL is derived from activated T cells. Also provided are vectors, host cells, antibodies, and recombinant methods for producing this human protein. The				

invention further relates to diagnostic and therapeutic methods useful for diagnosing and treating disorders related to this novel human protein, e.g. immune and autoimmune disorders.

RE.CNT 2 THERE ARE 2 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L8 ANSWER 5 OF 6 CAPLUS COPYRIGHT 2008 ACS on STN

AN 1997:757121 CAPLUS

DN 128:47297

OREF 128:9287a,9290a

TI Common gamma chain blocking agents

IN Burkly, Linda C.; Benjamin, Christopher D.; Hession, Catherine; Whitty, Adrian

PA Biogen, Inc., USA; Burkly, Linda C.; Benjamin, Christopher D.; Hession, Catherine; Whitty, Adrian

SO PCT Int. Appl., 111 pp.

CODEN: PIXXD2

DT Patent

LA English

FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 9743416	A1	19971120	WO 1997-US7870	19970509
W: AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE, DK, EE, ES, FI, GB, GE, GH, HU, IL, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, TJ, TM, TR, TT, UA, UG, US, UZ, VN, YU				
RW: GH, KE, LS, MW, SD, SZ, UG, AT, BE, CH, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, ML, MR, NE, SN, TD, TG				
CA 2253942	A1	19971120	CA 1997-2253942	19970509
AU 9729375	A	19971205	AU 1997-29375	19970509
EP 918858	A1	19990602	EP 1997-923611	19970509
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, FI				
US 6323027	B1	20011127	US 1998-189129	19981110
US 20020028202	A1	20020307	US 2001-824286	20010402
US 6770745	B2	20040803		
PRAI US 1996-17466P	P	19960510		
WO 1997-US7870	W	19970509		
US 1998-189129	A1	19981110		

AB The invention relates to antibodies which specifically bind to the gc chain of cytokine receptors, as well as to cell lines which produce such antibodies, pharmaceutical compns., and methods of treating immunol. diseases by treating patients with such antibodies. These cytokine receptor gc chain blocking antibodies or fragments are capable of blocking responses of a cell to a cytokine, e.g. interleukin 2, 4, 7, 9 and 15. The immunol. diseases include myasthenia gravis, inflammatory bowel disease, rheumatoid arthritis, lupus, multiple sclerosis, insulin-dependent diabetes, sympathetic ophthalmia, uveitis, allergy, asthma, parasitic disease, graft vs. host disease and psoriasis.

L8 ANSWER 6 OF 6 CAPLUS COPYRIGHT 2008 ACS on STN

AN 1997:400148 CAPLUS

DN 127:16508

OREF 127:3347a,3350a

TI T cell proliferation and anergy regulation by interleukins or other agents that stimulate cytokine receptor γ -chains

IN Boussiotis, Vassiliki A.; Nadler, Lee M.

PA Dana-Farber Cancer Institute, USA

SO PCT Int. Appl., 45 pp.

CODEN: PIXXD2

DT Patent

LA English

FAN.CNT 2

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
	-----	-----	-----	-----	-----
PI	WO 9717360	A2	19970515	WO 1996-US17927	19961112
	W: AU, CA, JP				
	RW: AT, BE, CH, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE				
	AU 9710506	A	19970529	AU 1997-10506	19961112
	AU 9947501	A	19991028	AU 1999-47501	19990910
PRAI	US 1995-556038	A	19951109		
	AU 1995-29152	A3	19950630		
	WO 1996-US17927	W	19961112		
AB	When stimulated through the T cell receptor(TCR)/CD3 complex without requisite costimulation through the CD28/B7 interaction, T cells enter a state of antigen specific unresponsiveness or anergy. This invention is based, at least in part, on the discovery that signaling through a common cytokine receptor γ chain (e.g., interleukin-2 receptor, interleukin-4 receptor, interleukin-7 receptor, interleukin-15 receptor) prevents the induction of T cell energy. This γ chain has been found to be associated with a JAK3 kinase having a mol. weight of about 116 kD (as determined by sodium dodecyl sulfate polyacrylamide gel electrophoresis) and signaling through the γ chain induces phosphorylation of the JAK3 kinase. Accordingly, methods for stimulating or inhibiting proliferation by a T cell which expresses a cytokine receptor γ chain are disclosed. In an example, induction of anergy in T cells was prevented by interleukin 2, 4, 7, and 15 by stimulation of a common .gamma.-chain of the interleukin receptors, and by phosphorylation of the JAK3 kinase.				